

09/978,146

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Term: 11 with L4

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|-----------|---|-------|-----------|
| <u>L5</u> | 11 with L4 | 5 | <u>L5</u> |
| <u>L4</u> | knockout or null adj mutation or gene near3 disrupt\$ | 20250 | <u>L4</u> |
| <u>L3</u> | 11 with L2 | 28 | <u>L3</u> |
| <u>L2</u> | transgen\$ | 34436 | <u>L2</u> |
| <u>L1</u> | pttg or ptsg | 78 | <u>L1</u> |

END OF SEARCH HISTORY

[Generate Collection](#)[Print](#)**Search Results - Record(s) 1 through 5 of 5 returned.**

-
- ☐ 1. [20030106080](#). 15 Oct 01. 05 Jun 03. [PTTG knockout](#) rodent as a model to study mechanisms for various physiological phenomena, including diabetes. Melmed, Shlomo, et al. 800/14; 435/353 435/354 800/18 A01K067/027 C12N005/06.
-
- ☐ 2. [20030068791](#). 20 Jul 01. 10 Apr 03. Manufacture of five-carbon sugars and sugar alcohols. Miasnikov, Andrei, et al. 435/158; 435/252.3 435/254.2 C12P007/18 C12N001/21 C12N001/18.
-
- ☐ 3. [20030017559](#). 30 Mar 01. 23 Jan 03. Method to produce succinic acid from raw hydrolysates. Donnelly, Mark I., et al. 435/145; 435/252.33 C12P007/46 C12N001/21.
-
- ☐ 4. [6743610](#). 30 Mar 01; 01 Jun 04. Method to produce succinic acid from raw hydrolysates. Donnelly; Mark I., et al. 435/145; 435/132 435/134 435/252.3 435/41. C12P007/46.
-
- ☐ 5. [6159738](#). 28 Apr 98; 12 Dec 00. Method for construction of bacterial strains with increased succinic acid production. Donnelly; Mark I., et al. 435/471; 435/140 435/145 435/252.3 435/252.31 435/252.33 435/252.9 435/440 435/472 536/23.1 536/23.2 536/23.7. C12P007/46 C12P007/00 C12N015/74 C12N015/00.
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| Terms | Documents |
|------------|-----------|
| L1 with L4 | 5 |

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09/978,146

=> d his

(FILE 'HOME' ENTERED AT 15:34:25 ON 05 OCT 2004)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 15:34:41 ON 05 OCT 2004

L1 772 S PTTG OR PTSG
L2 271926 S TRANSGEN?
L3 8 S L1(S)L2
L4 8 DUP REM L3 (0 DUPLICATES REMOVED)

=> d bib ab 1-8 l4

L4 ANSWER 1 OF 8 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 2004:307644 BIOSIS
DN PREV200400311233
TI Compositions and method for determining the presence of human PTTG peptide in a sample.
AU Melmed, Shlomo [Inventor, Reprint Author]; Pei, Lin [Inventor]
CS ASSIGNEE: Cedars-Sinai Medical Center
PI US 6750327 June 15, 2004
SO Official Gazette of the United States Patent and Trademark Office Patents, (June 15 2004) Vol. 1283, No. 3. <http://www.uspto.gov/web/menu/patdata.htm>
l. e-file.
ISSN: 0098-1133 (ISSN print).
DT Patent
LA English
ED Entered STN: 7 Jul 2004
Last Updated on STN: 7 Jul 2004
AB Polypeptides are expressed by the pituitary-tumor-transforming-gene (PTTG), formerly known as pituitary-tumor-specific-gene (PTSG), and nucleic acids encode them. Examples are the human and rat PTTG proteins. The nucleic acids may be applied to the production of a recombinant protein, and to the detection of the presence of PTTG genes in different species. The nucleic acids may be operatively linked to a vector, optionally provided with control and expression sequences and/or being carried by a host cell. The nucleic acids may also be delivered to a mammal to compensate for the absence, or a defective expression, of endogenous protein. The nucleic acids, proteins, and antibodies are also employed in diagnostic assays, as well as, for example, in the production of anti-PTTG antibodies (protein), therapeutic compositions and other applications of the proteins and antibodies. Various kits utilize nucleic acids, polypeptides, and/or antibodies. A **transgenic** non-human mammal expresses **PTTG**.

L4 ANSWER 2 OF 8 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 2004:257897 BIOSIS
DN PREV200400257955
TI Compositions and method for determining the presence of rat PTTG peptide in a sample.
AU Melmed, Shlomo [Inventor, Reprint Author]; Pei, Lin [Inventor]
CS Los Angeles, CA, USA
ASSIGNEE: Cedars-Sinai Medical Center
PI US 6723519 April 20, 2004
SO Official Gazette of the United States Patent and Trademark Office Patents, (Apr 20 2004) Vol. 1281, No. 3. <http://www.uspto.gov/web/menu/patdata.html>
. e-file.
ISSN: 0098-1133 (ISSN print).
DT Patent
LA English
ED Entered STN: 12 May 2004
Last Updated on STN: 12 May 2004
AB Polypeptides are expressed by the pituitary-tumor-transforming-gene (PTTG), formerly known as pituitary-tumor-specific-gene (PTSG), and

nucleic acids encode them. Examples are the human and rat PTTG proteins. The nucleic acids may be applied to the production of a recombinant protein, and to the detection of the presence of PTTG genes in different species. The nucleic acids may be operatively linked to a vector, optionally provided with control and expression sequences and/or being carried by a host cell. The nucleic acids may also be delivered to a mammal to compensate for the absence, or a defective expression, of endogenous protein. The nucleic acids, proteins, and antibodies are also employed in diagnostic assays, as well as, for example, in the production of anti-PTTG antibodies (protein), therapeutic compositions and other applications of the proteins and antibodies. Various kits utilize nucleic acids, polypeptides, and/or antibodies. A **transgenic** non-human mammal expresses **PTTG**.

L4 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2004 ACS on STN
 AN 2003:396993 CAPLUS
 DN 138:397254
 TI PTTG knockout rodent as a model to study mechanisms for various physiological phenomena, including diabetes
 IN Wang, Zhiyong; Melmed, Shlomo
 PA Cedars-Sinai Medical Center, USA
 SO PCT Int. Appl., 50 pp.
 CODEN: PIXXD2
 DT Patent
 LA English
 FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------|-----------------|--|----------|-----------------|----------|
| PI | WO 2003042356 | A2 | 20030522 | WO 2002-US30845 | 20020927 |
| | WO 2003042356 | A3 | 20031016 | | |
| | W: | AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | |
| | RW: | GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | |
| | US 2003106080 | A1 | 20030605 | US 2001-978146 | 20011015 |
| | EP 1435775 | A2 | 20040714 | EP 2002-773633 | 20020927 |
| | R: | AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK | | | |
| PRAI | US 2001-978146 | A | 20011015 | | |
| | WO 2002-US30845 | W | 20020927 | | |

AB The present invention discloses a null mutant (or knockout) rodent comprising in its germ cells an artificially induced PTTG null mutation. In some embodiments, the null mutant rodent can be generated by way of homologous recombination in an embryonic stem cell or germ cell. The inventive null mutant rodent can be used to study mammalian physiolo. at the cellular, tissue, and/or organismal level with respect to various phenotypes, including hyperglycemia, hypoinsulinemia, hypoleptinemia, diabetes, chromosomal aneuploidy, premature centromere division, chromosomal damage, aberrant mitotic cellular division, thrombocytopenia, thymic hyperplasia, splenic hypoplasia, testicular hypoplasia, and female subfertility. Also disclosed is an animal model for diabetes, a somatic or germ cell obtained from the null mutant rodent and a cell line derived from a cell obtained from the null mutant rodent.

L4 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2004 ACS on STN
 AN 2003:414081 CAPLUS
 DN 139:5775

TI **Transgenic** cells transfected with pituitary tumor transforming gene (**PTTG**) expression vectors and uses as cell model for study of **PTTG** and thyroglobulin expression
 IN Heaney, Anthony P.; Melmed, Shlomo
 PA USA
 SO U.S. Pat. Appl. Publ., 25 pp., Cont.-in-part of U.S. Ser. No. 854,326.
 CODEN: USXXCO
 DT Patent
 LA English
 FAN.CNT 13

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------|---|------|----------|-----------------|----------|
| PI | US 2003100530 | A1 | 20030529 | US 2002-264372 | 20021004 |
| | WO 9822587 | A2 | 19980528 | WO 1997-US21463 | 19971121 |
| | W: JP, US | | | | |
| | RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE | | | | |
| | US 6455305 | B1 | 20020924 | US 1999-894251 | 19990723 |
| | US 2003018001 | A1 | 20030123 | US 2000-730469 | 20001204 |
| | US 2002147162 | A1 | 20021010 | US 2001-777422 | 20010205 |
| | US 2003186902 | A1 | 20031002 | US 2001-854326 | 20010511 |
| | WO 2004033634 | A2 | 20040422 | WO 2003-US31393 | 20031003 |
| | WO 2004033634 | A3 | 20040715 | | |
| | W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | |
| | RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| PRAI | US 1996-31338P | P | 19961121 | | |
| | WO 1997-US21463 | W | 19971121 | | |
| | US 1999-894251 | A2 | 19990723 | | |
| | US 2000-569956 | A2 | 20000512 | | |
| | US 2000-687911 | A2 | 20001013 | | |
| | US 2000-730469 | A2 | 20001204 | | |
| | US 2001-777422 | A2 | 20010205 | | |
| | US 2001-854326 | A2 | 20010511 | | |
| | US 2002-264372 | A | 20021004 | | |

AB The present invention provides a TSH(TSH)-sensitive cell transfected with an expression vector comprising a DNA segment encoding a functional pituitary tumor transforming gene (PTTG) peptide, wherein the cell overexpresses PTTG in response to TSH. The nucleic acids of PTTG may be operatively linked to a vector, optionally provided with control and expression sequences and/or being carried by a host cell. Also disclosed is an in vitro cell model for the study of genetic regulation mediated by PTTG in a mammalian cell wherein PTTG expression can be modulated by exposing the cell to TSH or estrogen. In one embodiment, the cell model is used to study the effect of PTTG expression on sodium-iodide symporter (NIS) expression or to modulate NIS expression.

L4 ANSWER 5 OF 8 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
 AN 2002:608029 BIOSIS
 DN PREV200200608029
 TI Pituitary-tumor-transforming-genes, and related products.
 AU Melmed, Shlomo [Inventor, Reprint author]; Pei, Lin [Inventor]
 CS Los Angeles, CA, USA
 ASSIGNEE: Cedars-Sinai Medical Center
 PI US 6455305 September 24, 2002
 SO Official Gazette of the United States Patent and Trademark Office Patents, (Sep. 24, 2002) Vol. 1262, No. 4. <http://www.uspto.gov/web/menu/patdata.html>. e-file.

CODEN: OGUPE7. ISSN: 0098-1133.

DT Patent
LA English
ED Entered STN: 27 Nov 2002
Last Updated on STN: 27 Nov 2002
AB Polypeptides are expressed by the pituitary-tumor-transforming-gene (PTTG), formerly known as pituitary-tumor-specific-gene (PTSG), and nucleic acids encode them. Examples are the human and rat PTTG proteins. The nucleic acids may be applied to the production of a recombinant protein, and to the detection of the presence of PTTG genes in different species. The nucleic acids may be operatively linked to a vector, optionally provided with control and expression sequences and/or being carried by a host cell. The nucleic acids may also be delivered to a mammal to compensate for the absence, or a defective expression, of endogenous protein. The nucleic acids, proteins, and antibodies are also employed in diagnostic assays, as well as, for example, in the production of anti-PTTG antibodies (protein), therapeutic compositions and other applications of the proteins and antibodies. Various kits utilize nucleic acids, polypeptides, and/or antibodies. A **transgenic** non-human mammal expresses **PTTG**.

L4 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2004 ACS on STN

AN 2001:851361 CAPLUS

DN 136:622

TI Compositions and methods for modulating mammalian T-lymphocytes by targeted pituitary tumor transforming gene (PTTG) expression and/or function

IN Stoika, Rostyslav; Horwitz, Gregory A.; Zhang, Xun; Melmed, Shlomo

PA Cedars-Sinai Medical Center, USA

SO PCT Int. Appl., 185 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 13

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------|-----------------|--|----------|-----------------|----------|
| PI | WO 2001088116 | A2 | 20011122 | WO 2001-US15438 | 20010512 |
| | WO 2001088116 | A3 | 20020510 | | |
| | W: | AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | |
| | RW: | GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG | | | |
| | US 2003018001 | A1 | 20030123 | US 2000-730469 | 20001204 |
| | US 2002147162 | A1 | 20021010 | US 2001-777422 | 20010205 |
| | US 2003186902 | A1 | 20031002 | US 2001-854326 | 20010511 |
| | EP 1280907 | A2 | 20030205 | EP 2001-935431 | 20010512 |
| | R: | AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR | | | |
| | JP 2003533988 | T2 | 20031118 | JP 2001-585324 | 20010512 |
| PRAI | US 2000-569956 | A | 20000512 | | |
| | US 2000-687911 | A | 20001013 | | |
| | US 2000-730469 | A | 20001204 | | |
| | US 2001-777422 | A | 20010205 | | |
| | US 2001-854326 | A | 20010511 | | |
| | US 1996-31338P | P | 19961121 | | |
| | WO 1997-US21463 | W | 19971121 | | |
| | US 1999-894251 | A2 | 19990723 | | |
| | WO 2001-US15438 | W | 20010512 | | |

AB Disclosed is a method of inhibiting neoplastic cellular proliferation

and/or transformation of mammalian T-lymphocyte cells, including cells of human origin, in vitro or in vivo. Also disclosed are methods of immunomodulating, i.e., inhibiting or inducing, the activation of T-lymphocytes by modulating gene PTTG (pituitary tumor transforming gene) expression and/or gene PTTG1 protein function. In vitro methods for screening substances for new immunosuppressing or immunoenhancing agents that modulate the activation of mammalian T-lymphocytes are disclosed. Also disclosed are useful compns. and kits. CDNA for human gene PTTG1 has been cloned based on sequence homol. with the rat PTTG gene. The rat and human genes and their encoded proteins have been investigated, including their mRNA expression in tissues and cell lines, transactivation of gene transcription, effects of overexpression on cell proliferation and tumor induction, regulation of human bFGF secretion, and identification of a human PTTG gene family. Gene PTTG1 and its encoded protein have transforming activity, in vitro and in vivo, which requires a proline-rich domain in the polypeptide C-terminal region. The transforming protein encoded by gene PTTG1 may function through SH3-mediated signal transduction. Human gene PTTG1 mRNA is overexpressed in most cancers, including tumors of the colon, breast, ovary, and myeloid lineages. Gene PTTG1 mRNA expression also increases upon T cell activation by anti-CD3 antibodies or phytohemagglutinin (PHA) in parallel with T cell proliferation, after IL-2 mRNA induction, and before cyclophilin mRNA induction. Immunosuppressants hydrocortisone and cyclosporin A inhibit PHA-stimulated gene PTTG1 expression and T cell proliferation in normal T cells, while cyclosporin A and TGF- β 1 inhibit gene PTTG1 mRNA induction in activated leukemia cells. MRNA expression of gene PTTG1 is cell cycle-dependent in both T cells and a T cell leukemia line, with highest expression in G2/M-phase cells. Transfection of PHA-activated T cells with gene PTTG1 DNA encoding the C-terminal polypeptide region decreased the amount of S-phase cells and increased G2/M-phase cells.

L4 ANSWER 7 OF 8 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 2000:77834 SCISEARCH
GA The Genuine Article (R) Number: 276ET
TI Characterization of the murine pituitary tumor transforming gene (PTTG) and its promoter
AU Wang Z Y; Melmed S (Reprint)
CS UNIV CALIF LOS ANGELES, CEDARS SINAI MED CTR, CEDARS SINAI RES INST, SCH MED, 8700 BEVERLY BLVD, LOS ANGELES, CA 90048 (Reprint); UNIV CALIF LOS ANGELES, CEDARS SINAI MED CTR, CEDARS SINAI RES INST, SCH MED, LOS ANGELES, CA 90048
CYA USA
SO ENDOCRINOLOGY, (FEB 2000) Vol. 141, No. 2, pp. 763-771.
Publisher: ENDOCRINE SOC, 4350 EAST WEST HIGHWAY SUITE 500, BETHESDA, MD 20814-4110.
ISSN: 0013-7227.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 32
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB We recently isolated rat pituitary tumor transforming gene (PTTG) complementary DNA and showed its potent in vitro and in vivo transforming activity. We now characterize the mouse PTTG gene and its promoter. The entire gene is composed of five exons and four introns and spans about 7 kb. Northern analysis showed that PTTG was expressed in several tumor cell lines examined, but not in all normal tissues, implying a correlation between PTTG and tumorigenesis. Using rapid amplification of 5'-cDNA ends, the transcription start site was localized at -303 nucleotides upstream to the ATG codon in both F9 and AtT20 cells. An approximately 4.3-gb upstream region demonstrated promoter activity in AtT20 cells as well as other cell lines tested, and in vivo, the cloned promoter driving an enhanced green fluorescent protein

transgene exhibited transcriptional activation in testis and embryo. Serial deletions showed that -313 bp of the 5'-flanking region was critical for promoter activity. Three elements contribute to promoter activity. Both element A (-313/-293) and element C (-180/-160), in an electrophoretic mobility shift assay using NIH-3T3 nuclear extract, formed three specific complexes, which were competed by a known Spl oligo; one complex was supershifted by Spl antibody, and the other two complexes were both supershifted by an Sp3 antibody. Two mutants disrupting element A resulted in up to 70% loss of promoter activity and abrogated formation of specific DNA-protein binding complexes, implying a more important role for element A. Element B (-249/-229) shows more than 80% homology to a consensus c-myc element, but formed two specific complexes that differed from that of c-myc in the electrophoretic mobility shift assay. Thus, the integrity and possible cooperation among these elements contribute to the basal promoter activity of the mouse **PTTG** oncogene homolog.

L4 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2004 ACS on STN

AN 1998:352939 CAPLUS

DN 129:50520

TI Cloning and expression of mammalian pituitary tumor transforming gene (PTTG) and methods for detecting PTTG or its nucleic acid

IN Melmed, Shlomo; Pei, Lin

PA Cedars-Sinai Medical Center, USA; Melmed, Shlomo; Pei, Lin

SO PCT Int. Appl., 45 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 13

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|----|---|------|----------|-----------------|----------|
| | ----- | ---- | ----- | ----- | ----- |
| PI | WO 9822587 | A2 | 19980528 | WO 1997-US21463 | 19971121 |
| | W: JP, US | | | | |
| | RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE | | | | |
| EP | 944722 | A2 | 19990929 | EP 1997-953044 | 19971121 |
| | R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI | | | | |
| | JP 2002511734 | T2 | 20020416 | JP 1998-523945 | 19971121 |
| | US 6455305 | B1 | 20020924 | US 1999-894251 | 19990723 |
| | US 2003018001 | A1 | 20030123 | US 2000-730469 | 20001204 |
| | US 2002147162 | A1 | 20021010 | US 2001-777422 | 20010205 |
| | US 2003186902 | A1 | 20031002 | US 2001-854326 | 20010511 |
| | US 2002068716 | A1 | 20020606 | US 2001-949271 | 20010907 |
| | US 6723519 | B2 | 20040420 | | |
| | US 2002068353 | A1 | 20020606 | US 2001-949476 | 20010907 |
| | US 6750327 | B2 | 20040615 | | |
| | US 2002086845 | A1 | 20020704 | US 2001-949270 | 20010907 |
| | US 2002106778 | A1 | 20020808 | US 2001-949272 | 20010907 |
| | US 2003031662 | A1 | 20030213 | US 2002-136082 | 20020429 |
| | US 2003079242 | A1 | 20030424 | US 2002-136056 | 20020429 |
| | US 2003175266 | A1 | 20030918 | US 2002-135671 | 20020429 |
| | US 2003069197 | A1 | 20030410 | US 2002-163277 | 20020604 |
| | US 2003167496 | A1 | 20030904 | US 2002-176812 | 20020621 |
| | US 2003177511 | A1 | 20030918 | US 2002-176549 | 20020621 |
| | US 2003114378 | A1 | 20030619 | US 2002-261717 | 20020930 |
| | US 2003147892 | A1 | 20030807 | US 2002-261821 | 20020930 |
| | US 2003148977 | A1 | 20030807 | US 2002-262258 | 20020930 |
| | US 2003148978 | A1 | 20030807 | US 2002-262264 | 20020930 |
| | US 2003153522 | A1 | 20030814 | US 2002-261787 | 20020930 |
| | US 2003152573 | A1 | 20030814 | US 2002-262252 | 20020930 |
| | US 2003100530 | A1 | 20030529 | US 2002-264372 | 20021004 |
| | US 2003131366 | A1 | 20030710 | US 2002-283797 | 20021029 |
| | US 2003130219 | A1 | 20030710 | US 2002-284126 | 20021029 |
| | US 2003140359 | A1 | 20030724 | US 2002-283771 | 20021029 |
| | US 2003186910 | A1 | 20031002 | US 2002-283874 | 20021029 |

| | | | |
|------|-----------------|----|----------|
| PRAI | US 1996-31338P | P | 19961121 |
| | US 1997-65825P | P | 19971114 |
| | WO 1997-US21463 | W | 19971121 |
| | US 1999-894251 | A2 | 19990723 |
| | US 2000-569956 | A2 | 20000512 |
| | US 2000-687911 | A2 | 20001013 |
| | US 2000-730469 | A2 | 20001204 |
| | US 2001-777422 | A2 | 20010205 |
| | US 2001-854326 | A2 | 20010511 |

AB Polypeptides encoded by the pituitary tumor transforming gene (PTTG), formerly known as pituitary tumor specific gene (PTSG) are disclosed. PTTG nucleic acids may be applied to the production of a recombinant protein and to the detection of the presence of PTTG genes in different species. The nucleic acids, proteins, and antibodies may be employed in diagnostic assays, as well as, for example, in the production of anti-PTTG antibodies and therapeutic compns.. The nucleic acids may also be delivered to a mammal to compensate for the absence, or a defective expression, of endogenous protein. PTTG was identified in a rat pituitary tumor cell cDNA library by differential display PCR. Both human and rat PTTG cDNAs were cloned. PTTG was strongly expressed in testis and in carcinoma cells. Recombinant 3T3 cells expressing PTTG caused tumor formation in mice.

=>